

beneficial effects of aprotinin use probably occur even before the carrying out of cardiopulmonary bypass, but its dramatic effect in these patients has meant that most investigators have focused their research efforts on that procedure. In noncardiac surgical procedures such as described in this report, trauma to scar tissue may release tissue plasminogen activator and activate fibrinolysis that, in turn, is attenuated by aprotinin. Unfortunately, no markers of hemostasis such as fibrin-degradation products were evaluated in this patient. Future controlled trials will offer the opportunity to evaluate the mechanism of action of aprotinin in noncardiac surgical procedures.

The case reported here may represent two possible uses of aprotinin that deserve further investigation: minimizing blood loss during an orthopedic operation and minimizing blood loss in HIV-positive patients requiring surgical therapy. The results also suggest that aprotinin may have beneficial effects in many surgical procedures not yet studied. The well-known dangers of bleeding during surgical procedures and of transfusion therapy make this an important subject worthy of further evaluation. The apparent safety of aprotinin administration means that the drug, if used in a wide variety of procedures, could have wide surgical application.

#### REFERENCES

1. Royston D, Bidstrup BP, Taylor KM, Sapsford RN: Effect of aprotinin on the need for blood transfusion after repeat open-heart surgery. *Lancet* 1987; 2:1289-1291
2. Bidstrup BP, Royston D, Taylor KM, Sapsford RN: Reduction in blood loss and blood use after cardiopulmonary bypass with high dose aprotinin (Trasylol). *J Thorac Cardiovasc Surg* 1989; 97:364-372
3. Bidstrup BP, Royston D, McGuinness C, et al: Aprotinin in aspirin treated patients. *Perfusion* 1990; 5:77-81
4. Alajmo F, Calamai G, Perna AM, et al: High-dose aprotinin: Hemostatic effects in open heart operations. *Ann Thorac Surg* 1989; 48:536-539
5. Dietrich W, Barankay A, Diltthey G, et al: Reduction of homologous blood requirement in cardiac surgery by intraoperative aprotinin application—Clinical experience in 152 cardiac surgical patients. *Thorac Cardiovasc Surg* 1989; 37: 92-98
6. Fraedrich G, Weber C, Bernard C, Hettwer A, Schlosser V: Reduction of blood transfusion requirement in open heart surgery by administration of high doses of aprotinin—Preliminary results. *Thorac Cardiovasc Surg* 1989; 37:89-91
7. Royston D: High-dose aprotinin therapy: A review of the first five years' experience. *J Cardiothorac Vasc Anesth* 1992; 6:76-100
8. van Oeveren W, Jansen NJG, Bidstrup BP, et al: Effects of aprotinin on hemostatic mechanisms during cardiopulmonary bypass. *Ann Thorac Surg* 1987; 44:640-645
9. Dietrich W, Spannagl M, Jochum M, et al: Influence of high-dose aprotinin treatment on blood loss and coagulation patterns in patients undergoing myocardial revascularization. *Anesthesiology* 1990; 73:1119-1126
10. Kang Y, De Wolf AM, Aggarwal S, Campbell E, Martin LK: In vitro study of the effects of aprotinin on coagulation during orthotopic liver transplantation. *Transplant Proc* 1991; 23:1934-1935
11. Himmelreich G, Kierzek B, Neuhaus P, Slamer KJ, Riess H: Fibrinolytic changes and the influence of the early perfusate in orthotopic liver transplantation with intraoperative aprotinin treatment. *Transplant Proc* 1991; 23:1936-1937
12. Mallett S, Rolles K, Cox D, Burroughs A, Hunt B: Intraoperative use of aprotinin (Trasylol) in orthotopic liver transplantation. *Transplant Proc* 1991; 23:1931-1932
13. Cottam S, Hunt B, Segal H, Ginsburg R, Potter D: Aprotinin inhibits tissue plasminogen activator-mediated fibrinolysis during orthotopic liver transplantation. *Transplant Proc* 1991; 23:1933
14. Royston D: The serine antiprotease aprotinin (Trasylol): A novel approach to reducing postoperative bleeding. *Blood Coag Fibrinol* 1990; 1:55-69
15. Ketterl R, Haas S, Heiss A, et al: Zur Wirkung des natürlichen Proteinaseinhibitoren Aprotinin auf die Plättchenfunktion beim alloarthoplastischen Hüftgelenkersatz. *Med Welt* 1982; 33:480-486
16. Beck OJ, Oeckler R: Subarachnoid haemorrhage. *Br Med J* 1982; 284:1050
17. Beck OJ, Oeckler R: Frühdiagnose und Therapie der Aneurysma blutungen. *Munch Med Wochenschr* 1981; 123:561-564
18. Tzonos T, Giromini D: Aprotinin for intraoperative haemostasis. *Neurosurg Rev* 1981; 4:193-194
19. Kösters S, Wand H: Über die Beeinflussung des Blutverlustes nach Prostataoperationen durch prä-öperative Applikation von Antifibrinolytika. *Urologie* 1973; 12:295-296

## Successful Enteral Refeeding After Massive Small Bowel Resection

DONNA J. RODRIGUEZ, RD, CNSD  
FREDERICK W. CLEVINGER, MD  
Albuquerque, New Mexico

EXTENSIVE SMALL BOWEL RESECTION (>70%) is associated with severe nutritional consequences. The short-gut syndrome usually develops in patients with less than 150 cm of intact small bowel.<sup>1</sup> Malabsorption, dehydration, metabolic abnormalities, and nutrient deficiencies are possible problems if nutrition support is not implemented immediately after bowel resection.

Total parenteral nutrition (TPN), the most aggressive form of nutrition support, is usually necessary in the initial stages of recovery. The enteral route is preferred for long-term support, but tolerance is dependent on the extent of the small bowel resection, the site of remaining bowel (especially the ileum and ileocecal valve), the functional capacity of the remaining gut, and the presence or absence of concomitant gastrointestinal disease.<sup>2</sup> The amount of small bowel necessary for adequate nutrient delivery by the enteral route has been reported at variable lengths.

### Report of a Case

The patient, a 62-year-old man, was admitted to the Level I Trauma Center at the University of New Mexico Medical Center (Albuquerque) with multiple stab wounds to his back and neck following a family altercation. Although enteral nutrition support (tube feeding) was started on the first day of hospital admission, TPN was instituted on day 5 because of tube feeding intolerance (diarrhea, abdominal distention). An exploratory laparotomy was done on hospital day 16 to rule out an intra-abdominal source for the patient's sepsis and abdominal distention, following which infarcted small bowel was resected from the proximal jejunum to the ileum. The distal ileum (57 cm in length), the ileocecal valve, and the colon were left intact.

After this operation, TPN was continued in amounts to meet the patient's estimated calorie and protein needs

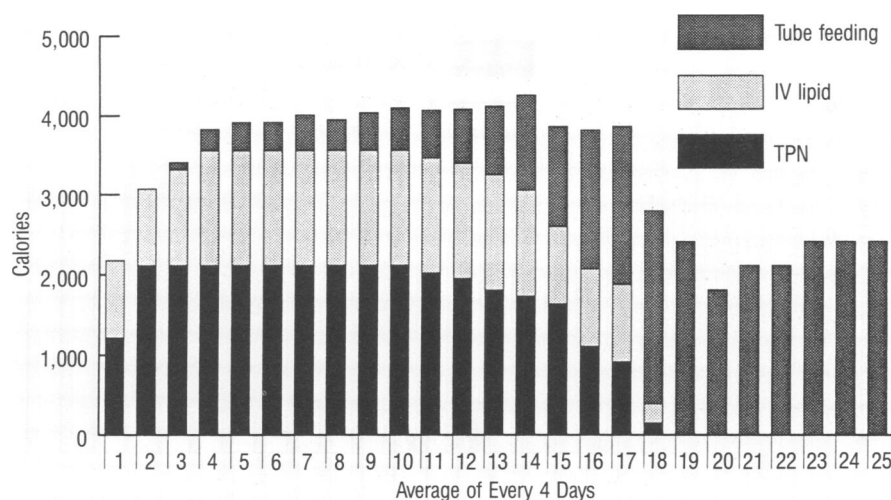
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From the Surgical Nutrition Support Services (Ms Rodriguez and Dr Clevenger) and the Division of Burns and Trauma (Dr Clevenger), Department of Surgery, University of New Mexico School of Medicine, Albuquerque.

Reprint requests to Donna J. Rodriguez, RD, CNSD, Surgical Nutrition Support Service, Department of Surgery, University of New Mexico School of Medicine, Albuquerque, NM 87131-5341.



**Figure 1.**—The graph shows the patient's calorie intake by 3 different nutrition support delivery routes over an average of every 4 days after massive small bowel resection. IV = Intravenous, TPN = Total parenteral nutrition

until postoperative day 12 when the administration of the isotonic, peptide-based formula, Peptamen (Clintec Nutrition, Deerfield, Illinois), was started at 15 ml per hour through a small-bore feeding tube. This nutrition support regimen was continued for the next 20 days, at which time the tube feeding rate was increased by 5 ml per hour and then by 5 to 10 ml per hour on postoperative days 39, 46, 49, 52, 56, 60, 62, 63, 68, and 69. The parenteral nutrition and intravenous lipid rates were tapered accordingly until the tube feeding goal of administering Peptamen at 100 ml per hour was reached (Figure 1). Formed stools were noted after each tube feeding rate increase. The patient remained in positive nitrogen balance (+1, +2.2, +7, +4.7)\* during the tube feeding rate progression (Figure 2). As more solid foods were tolerated, the tube feeding rate was also tapered accordingly.

When discharged on hospital day 112, the patient was consuming a low-fat, low-residue diet in six small feedings with oral formula supplements. Although serum albumin levels had decreased from 32 to 17 grams per liter (3.2 to 1.7 grams per dl) during the patient's septic episode, he was discharged with a serum albumin level of 30 grams per liter. His admission weight (59.2 kg) remained stable at the time of discharge (58.9 kg; see Figure 2).

The patient's follow-up visit two months after discharge showed that he was maintaining a good nutritional state. His weight (56.4 kg) was essentially stable. His visceral protein status had continued to improve, the serum albumin level being 40 grams per liter. Serum electrolyte, mineral, trace elements, and iron levels were all within normal limits.

\*Nitrogen balances were calculated by subtracting 24-hour nitrogen losses (urine, insensible) from 24-hour nitrogen intake. The following equation was used:

$$[\text{protein intake (grams/24hr)}] + 6.25 - \\ [24\text{-hr urine for urea nitrogen (grams)} + \\ 4 \text{ (insensible losses)}]$$

A positive nitrogen balance indicates an anabolic state; a negative nitrogen balance indicates a catabolic state.

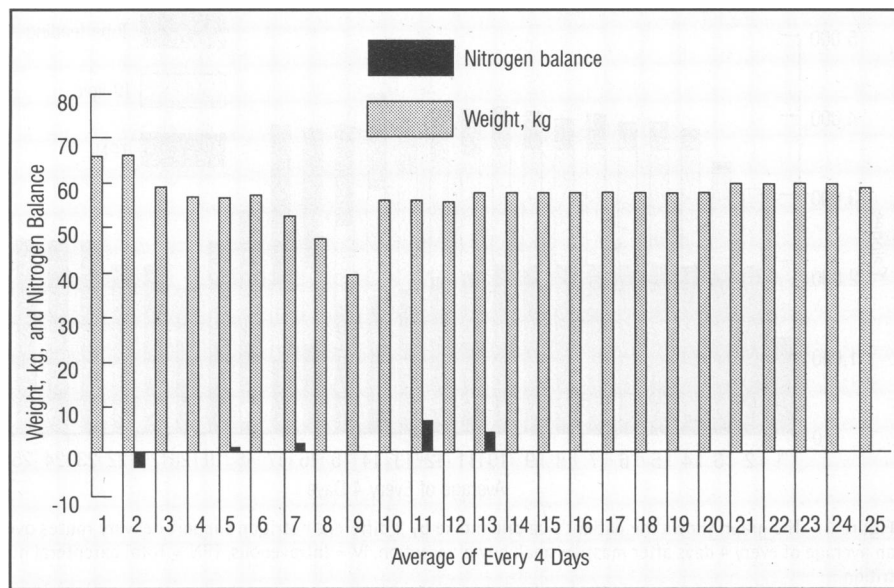
## Discussion

After small bowel resection, intestinal adaptation occurs gradually, requiring more than a year to reach its maximum absorptive capacity. Hyperplasia of the microvilli, lengthening and dilation of the small bowel, and increased brush border enzyme activity all contribute to the increased surface area and absorptive capacity of the short gut.<sup>3</sup>

The delivery of enteral nutrients is the most important stimulus for mucosal growth and adaptation.<sup>2</sup> Numerous studies have been done to clarify the effects of different factors. Continuous enteral feedings of small volumes are more effective than intermittent doses of larger volumes. The flow of bile and pancreatic secretions stimulate mucosal growth distal to the point of delivery.<sup>3</sup> Secretin, cholecystokinin, glucagon, and enteroglucagon have all been suggested as trophic hormones for the gastrointestinal tract. Brush border enzyme activity increases according to the composition of the enteral feeding; more specifically, peptidases are stimulated by protein, disaccharidases by carbohydrate.<sup>2</sup>

Administering enteral feedings in the "elemental" form was originally favored for patients with impaired digestion and absorption.<sup>4</sup> The earliest elemental formulas contained protein as free amino acids, carbohydrate as glucose, and minimal quantities of fat. It was thought that providing nutrients in these forms facilitated maximal absorption. Clinical experience with these formulas has demonstrated frequent patient intolerance. The nausea, vomiting, and diarrhea that occurred were presumably due to formula hypertonicity—range, 500 to 910 mOsm per kg. Substantially diluted formulas were better tolerated, but their use resulted in inadequate nutritional intakes.

In comparison, accumulating information now suggests that peptide-based formulas have several advantages in patients with gastrointestinal compromise. Because different uptake carrier systems are used for amino



**Figure 2.**—The patient's nutritional status is demonstrated by nitrogen balance and weights over an average of every 4 days after small bowel resection.

acids and peptides, providing protein in both forms results in more rapid and efficient absorption.<sup>4,5</sup> Improved nitrogen use and retention and the maintenance of visceral protein status have been reported when the use of peptide-based formulas was compared with that of elemental diets.<sup>5</sup> A decreased incidence of diarrhea has also been noted with peptide-based formulas, due to presumably better absorption and low osmolalities. In addition, available data suggest that peptides stimulate a greater release of gut hormones and growth factors than do free amino

acids, a particular advantage for patients with the short-gut syndrome.

#### REFERENCES

1. Beyer PL, Frankenfield DC: Enteral nutrition in extreme short bowel. *Nutr Clin Pract* 1987; 2:60-64
2. Purdum PP 3d, Kirby DF: Short-bowel syndrome: A review of the role of nutrition support. *JPEN J Parenter Enteral Nutr* 1991; 15:93-101
3. Deitel M, Wong KH: The short bowel syndrome, *In* Deitel M (Ed): *Nutrition in Clinical Surgery*. Philadelphia, Pa, Williams & Wilkins, 1985, pp 255-275
4. Zaloga GP: Physiologic effects of peptide-based enteral formulas. *Nutr Clin Pract* 1990; 5:235-237
5. Brinson RR, Hanumanthu SK, Pitts WM: A reappraisal of the peptide-based enteral formulas: Clinical applications. *Nutr Clin Pract* 1989; 4:211-217